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EXAMINER	
FORD, ALLISON M	

ART UNIT	PAPER NUMBER
1651	

DATE MAILED: 11/29/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/802,955	MICHAL ET AL.	
	Examiner	Art Unit	
	Allison M. Ford	1651	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *17 October 2005*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,3,5-8 and 19-22* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,3,5-8 and 19-22* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *17 October 2005* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Applicant's amendments filed 17 October 2005 to claims 1, 3, 5, 7, 8 and 19-22 have been entered. Claims 2, 4, 9-18, and 23-62 have been cancelled. Claims 1, 3, 5, 6, 7, 8 and 19-22 remain pending in the current application, all of which have been considered on the merits.

Priority

Acknowledgement is made of applicant's claim for status as a CIP of copending application 10/414,602, filed 04/15/2003.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1, 3, 5-8, and 19-22 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Applicant's claim 1 is directed to a method, comprising identifying an infarct region within the ventricle of a human subject; and delivering a non-antigenic cell line comprising alpha-1,3-galactosyltransferase (GGTA1) knock-out swine cells to the infarct region within the ventricle of the human subject. Claim 3 requires both chromosomal copies of a gene for alpha-1,3-galactosyltransferase (GGTA1) knock-out swine cells have been disrupted. Claim 7 requires the delivery of the non-antigenic cell line to occur within 2 weeks of a myocardial infarction (MI).

Claim 1 is indefinite because it is not clear how one delivers a cell line; rather it would be more appropriate to require delivery of cells from a specific cell line. Additionally, the phrase "cell line

comprising alpha-1,3-galactosyltransferase knock-out swine cells" is confusing, as the term 'comprising' is open language and infers inclusion of additional components, such as additional cell types; however, a cell line would consist of only a single cell type. In the instant case a non-antigenic cell line of alpha-1,3-galactosyltransferase knock-out swine cells would only consist of alpha-1,3-galactosyltransferase knock-out swine cells. It appears it would be more appropriate to require delivery of non-antigenic alpha-1,3-galactosyltranserfase (GGTA1) knock-out swine cells to the infarct region within the ventricle of the human subject.

Claim 3 is indefinite because a gene does not encode for GGTA1 *knock-out swine cells*; it appears applicant intends to require both chromosomal copies of the gene for alpha-1,3-galactosyltransferase (GGTA1) to have been disrupted. However, even upon this interpretation the claim remains indefinite, as it is unclear if it further limits the parent claim 1. Claim 1 requires the cells to be alpha-1,3-galactosyltransferase (GGTA1) *knock-out* swine cells, inferring the alpha-1,3-galactosyltransferase genes are inherently disrupted in the cell line; therefore the limitation of claim 3 does not further limit claim 1.

Claims 7 and 8 are indefinite because, as in claim 1, the method does not deliver a cell line, per se, but rather cells from a cell line. Additionally, claim 8 would be clearer if it read, "...wherein the non-antigenic cells carry a nucleic acid encoding a detectable polypeptide operably linked to a promoter." Also note there is a typographical error placing two periods (".") at the end of claim 8, obviously only one is needed; appropriate correction is requested.

Applicant's claim 19 is directed to a method, comprising identifying an infarct region within a ventricle of a subject; applying a pacing therapy to the ventricle to pre-excite the infarct region to unload the infarct region from mechanical stress; and delivering at least one structurally reinforcing component to the infarct region after application of the pacing therapy. Claim 22 requires the method of claim 19 to further comprise modifying the pacing therapy based upon sensor measurements.

First it is noted that the article "an" is inappropriate at the fourth line of claim 19 "delivering ~~an~~ at least one structurally reinforcing component". Second, the phrase "unload the infarct region from mechanical stress" is unclear.

Claim 22 is indefinite because no steps are claimed for described for taking sensor measurements, it is not clear what is being measured by what sensor, and it is not clear what modifications are made, or how they are made, based upon the undisclosed sensor measurements.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 1, 3, and 5-8 are rejected under 35 U.S.C. 103(a) as being unpatentable over Etzion et al (J Mol Cell Cardiol, 2001), in view of Dinsmore (US Patent 5,979,449) and Gustafsson et al (US Patent 6,153,428).

Applicant's claim 1 is directed to a method, comprising identifying an infarct region within the ventricle of a human subject; and delivering a non-antigenic cell line comprising alpha-1,3-galactosyltransferase (GGTA1) knock-out swine cells to the infarct region within the ventricle of the human subject. Claim 3 requires both chromosomal copies of a gene for alpha-1,3-galactosyltransferase (GGTA1) knock-out swine cells have been disrupted. Claim 5 requires the method of claim 1 to further comprise delivering at least one structurally reinforcing agent to the infarct region to increase the modulus of elasticity of the infarct region. Claim 6 requires the cells to replace damaged cells in and around the infarct region. Claim 7 requires the delivery of the non-antigenic cell line to occur within 2 weeks of a

myocardial infarction (MI). Claim 8 requires the non-antigenic cells to carry a nucleic acid encoding a detectable polypeptide operably linked to a promoter.

Etzion et al teach inducing myocardial infarction in the ventricle of rat subjects; identifying the infarct region visually on the basis of a surface scar and wall motion akinesis; and delivering embryonic rat cardiomyocytes (which applicant calls a structurally reinforcing agent) to the infarct region (See Pg. 1322). Cell transplantations were performed seven days after myocardial infarction (See Pg. 1322, col. 2) (Claim 7). Representative samples of the transplanted cells were transfected with recombinant adenovirus carrying the nuclear reporter gene *LacZ*, encoding for β-galactosidase, X-gal staining revealed blue nuclei, indicating that the *LacZ* gene was expressed; the *LacZ* must be operably linked to an operator for proper expression; therefore the nucleic acid encoding the detectable β-galactosidase was operably linked to a promoter (See Pg. 1324, col. 2 and Fig. 1(d) & Lewin Pg. 277-280) (Claim 8).

Though Etzion et al teach the transplanted myocardiocytes engraft into and are able to survive in the infarcted myocardium and increase wall thickness and reduce wall stress, they do not specifically state the engrafted cells replace damaged cells in and around the infarct region or that the cells increase the modulus of elasticity of the infarct region. However, because Etzion et al teach the same process of delivering cells to an infarct region of a subject, as claimed in the current application, the resulting infarct region is one and the same as in the current application, and thus the engrafted cells replace the damaged cells in and around the infarct region and increase the modulus of elasticity of the infarct region the same as in the current application (Claims 5 and 6).

Etzion et al use rat models to test the effectiveness of cardiomyocyte transplantation as a treatment of ischemic heart disease; however, due to the prevalence of heart disease, particularly ischemic-related heart disease, among humans in the U.S. (40% of total mortality related to heart disease, and 60-75% of heart disease deaths related to ischemic heart disease (See Dinsmore, col. 1, ln 6-22)) it would have been obvious to one of ordinary skill in the art, at the time the invention was made, to utilize

the cell transplant treatment of Etzion et al in human patients. Though Etzion et al perform syngeneic transplants between rats, the limited supply of human donors makes syngeneic transplants in humans more difficult. However, at the time the invention was made, swine were known to be the preferred tissue donor source for xenotransplantations to humans when syngeneic human tissue was unavailable, due to greater availability of swine tissue compared to human tissue, similarity in organ size between the tissues, reduced risk of zoonotic infections and reduced ethical concerns (See, e.g. Gustafsson et al, col. 1 & Dinsmore, col. 1-2). However, in xenotransplantations immunorejection due to species-specific antigens is a major concern; therefore, in order to reduce the risk of immunorejection of swine cardiomyocytes it would have been obvious to one of ordinary skill in the art to use non-antigenic swine cardiomyocytes for xenotransplantation. Such non-antigenic swine cells, including swine cardiomyocytes were known at the time of the invention, for example Gustafsson et al teach transgenic α-1,3-galactosyltransferase (GGTA1) knock-out swine cells, that are immunogenically tolerable to recipients, including human patients in the case of xenotransplantations (See Gustafsson et al col. 2, ln 19-33). In the transgenic α-1,3-galactosyltransferase (GGTA1) knock-out swine cells the genes, on one or both alleles, has been disrupted to reduce the amount of α-1,3-galactosyltransferase produced to an extent sufficient to prevent the cells' ability to provide carbohydrates with the Galα1-3Galβ1-4GlcNAc epitope from being provided to the cell surface, thereby rendering the cells immunogenically tolerable to the intended recipient, especially humans. Whole animals or individual cells and/or tissues of any desired type can be produced in the GGTA1 knock-out phenotype, with one or both alleles having been disrupted (See Gustafsson et al, col. 6, ln 2-5) (Claim 3). Gustafsson et al teach the transgenic GGTA1 knock-out cells can be used as a source of cells for xenotransplantation (See Gustafsson et al, col. 5, ln 58-col. 6, ln 9).

Therefore, at the time the invention was made it would have been obvious to one of ordinary skill in the art to perform the cell transplantation treatment of Etzion et al on human patients experiencing ischemic heart disease using donor GGTA1 knock-out swine cardiomyocytes. One of ordinary skill in the

art would have been motivated to perform the cell transplantation treatment of Etzion et al on human patients in order to repair the infarct region of the ventricle subject to ischemia; one would have been motivated to use swine cells because, next to syngeneic human cells, swine cells are recognized as the preferred cell type for xenotransplantation into human recipients; particularly one would have been motivated to use the GGTA1 knock-out swine cardiomyocytes of Gustafsson et al in order to reduce the possibility of immunorejection. One would have expected success using GGTA1 knock-out swine cardiomyocytes as the type of donor swine cell because they retain the same functional properties as normal swine cardiomyocytes, but lack the antigenic epitope; one would have expected success performing the xenotransplantation of swine cells to human patients because swine tissues and cells are known to be particularly suitable for human use (See Dinsmore and Gustafsson et al); and finally one would have expected that transplantation of the GGTA knock-out swine cardiomyocytes to the infarct region of a human suffering from ischemic heart disease would successfully treat the ischemia because Etzion et al teach transplantation of exogenous cardiomyocytes into an infarct region of a ventricle results in increased wall thickness and decreased wall stress.

Therefore the invention as a whole would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made.

Claims 19-22 are rejected under 35 U.S.C. 103(a) as being unpatentable over Salo (US 2003/0105493 A1), in view of Etzion et al (J Mol Cell Cardiol, 2001), and further in view of Gustafsson et al (US Patent 6,153,428).

Applicant's claim 19 is directed to a method, comprising identifying an infarct region within a ventricle of a subject; applying a pacing therapy to the ventricle to pre-excite the infarct region to unload the infarct region from mechanical stress; and delivering at least one structurally reinforcing component to the infarct region after application of the pacing therapy. Claim 20 requires the at least one structurally

reinforcing component to comprise α-1,3-galactosyltransferase (GGTA1) knock-out swine cells. Claim 21 requires the pacing therapy to comprise a bradycardia pacing algorithm. Claim 22 requires the method of claim 19 to further comprise modifying the pacing therapy based upon sensor measurements.

Various methods were known at the time of the current invention on how to treat myocardial infarctions, caused by ischemia; some methods focused on reducing the ventricular remodeling that follows myocardial infarction, while other methods focus on repairing the infarcted zone before severe ventricular remodeling took place.

For example, Salo teaches a method for minimizing the ventricular remodeling that normally follows a myocardial infarction, comprising identifying an infarct region within a ventricle of a subject by any of a number of means, including ultrasonic imaging, PET scans, thallium scans, and MRI perfusion scans (See Salo, Pg. 2, paragraph 0016); and applying a pacing therapy to sites in proximity to the infarct to pre-excite the infarct region in a manner that lessens the mechanical stress to which the infarct region is subjected, thus reducing the stimulus for remodeling (See Salo, Pg. 1, paragraph 0006). Salo teaches the pacing therapy can comprise a bradycardia pacing algorithm, such as an inhibited demand mode or a triggered mode (See Salo, Pg. 2, paragraph 0015 & Pg. 3, paragraph 0020) (Claim 21). Salo teach that pacemakers used for such pacing therapies includes sensors and controller means to modify the pacing pulses in response to changing readings from the sensors (See Salo, Pg. 3, paragraph 00017) (Claim 22). Salo teaches that pre-exciting the infarct region results in a decreased preload and after load, which decreases the mechanical stress experienced by the region; thereby lessening the metabolic demands of the region and can effectively prevent or minimize the infarct remodeling (See Salo Pg. 2, paragraphs 0012-0014).

Alternatively, Etzion et al teach a method for replacing the damaged myocardial tissue with healthy tissue, their method comprises identifying the infarct region visually on the basis of a surface scar and wall motion akinesis; and delivering exogenous embryonic cardiomyocytes to the infarct region (See

Pg. 1322). Etzion et al teach the transplanted myocardiocytes engraft into and are able to survive in the infarcted myocardium and increase wall thickness and reduce wall stress. Etzion et al teach performing allogeneic transplants from embryonic rats to adult rats; however, even in their syngeneic transplantations immune reactions caused rejection problems (See Pg. 1329, col. 1). Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to use cells or cell lines that are immunogenically tolerable to the recipient, so as to not elicit an immune reaction. Gustafsson et al teach such a cell line, transgenic α-1,3-galactosyltransferase (GGTA1) knock-out swine cells, that is immunogenically tolerable to recipients, even in xenotransplantations (See Gustafsson et al col. 2, ln 19-33). In the transgenic α-1,3-galactosyltransferase (GGTA1) knock-out swine cells the genes, on one or both alleles, has been disrupted to reduce the amount of α-1,3-galactosyltransferase produced to an extent sufficient to prevent the cells' ability to provide carbohydrates with the Galα1-3Galβ1-4GlcNAc epitope from being provided to the cell surface, thereby rendering the cells immunogenically tolerable to the intended recipient. Whole animals or individual cells and/or tissues of any desired type can be produced in the GGTA1 knock-out phenotype, with one or both alleles having been disrupted. Gustafsson et al teach the transgenic GGTA1 knock-out cells can be used as a source of cells for transplantation (See Gustafsson et al, col. 5, ln 58-col. 6, ln 9). Therefore, one of ordinary skill in the art, at the time the invention was made, would have been motivated to use GGTA1 knock-out myocardiocyte cells, taught by Gustafsson et al, in the method of Etzion et al, in order to prevent immune rejection. One of ordinary skill in the art would have been especially motivated to use the GGTA1 knock-out myocardiocytes of Gustafsson et al in the method of Etzion et al, if the cells were being delivered to a human subject as treatment of a myocardial infarction because Gustafsson et al teach the Galα1-3Galβ1-4GlcNAc epitope is the major target for anti-swine xenoreactive human natural antibodies (See Gustafsson et al, col. 1, ln 38-44). One would have expected success using swine GGTA1 knock-out myocardiocytes, developed by Gustafsson et al, in the method of Etzion et al because Etzion et al teach successfully transplanting the

embryonic myocardiocytes into the infarct region in the ventricle of a subject; however Etzion et al did report problems associated with immune reaction from the allogeneic cells. Using the immunogenically tolerable GGTA1 knock-out swine cells of Gustafsson et al, one would expect elimination of the immune reaction, and overall success of the transplant.

At the time the invention was made it would have been obvious to one of ordinary skill in the art to combine the two different treatments described above to provide superior therapy to patients with myocardial infarction due to ischemia. It would have been obvious to first apply the pacing therapy of Salo, such as the bradycardia pacing algorithm, in order to decrease the mechanical stress experienced by the region, thereby effectively minimizing the degree of infarct remodeling; then after the infarct region is stabilized in size, apply the cell transplant treatment of Etzion et al, using the non-antigenic GGTA1 knock-out swine cells of Gustafsson et al, to repair and replace damaged tissue in and around the infarction (Claims 19 and 20). One of ordinary skill in the art would have been motivated to combine the two therapies because both were known at the time of the invention to be effective treatments for myocardial infarction. A combination of two sequential treatments, each known for treatment of different aspects of the same disease, with no exception for negative interaction between the two treatments, would be obvious to one of ordinary skill in the art. The idea of first minimizing the damaged area, and then replacing the damaged area with healthy tissue, when methods of performing both steps are known, would be obvious; motivation for combining the treatments would come from the desire to provide the most effective treatment to repair and regenerate the myocardial tissue following ischemia. One would expect success combining the known treatments because Salo and Etzion et al each teach success with their treatments individually, and there is no evidence or reason to believe that the combined treatments would produce negative interactions. Therefore the invention as a whole would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made.

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and, *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 1, 5 and 7 stand provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1, 2, and 4 of copending Application No. 10/414,602. Although the claims of copending Application 10/414,602 are not identical, they are not patentably distinct from the current application because current claims 1, 5 and 7 anticipate copending claims 1, 2, and 4, respectively. Though the current claim 1 describes a specific cell line, GGTA1 knock-out swine cells, the cells read on a "structurally reinforcing agent" as described in the copending application.

Additionally, claims 1 and 5 stand provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 2 and 3 of copending Application No. 10/414,767. Although the claims of copending Application 10/414,767 are not identical, they are not patentably distinct from the current application because it would have been obvious to one of ordinary skill in the art to deliver an implant comprising cells as the solid material to the infarct region for the purpose of reinforcing the weakened infarct region and increasing the compliance of the ventricle. One of ordinary skill in the art would have been motivated to deliver an implant comprising cells in order to rebuild and strengthen the weakened area, the cells provided in the implant would then be expected to develop into new tissue to rebuild the region.

These are provisional obviousness-type double patenting rejections because the conflicting claims have not in fact been patented.

Response to Arguments

Applicants arguments received in the reply filed 17 October 2005 have been fully considered. Applicant's arguments are directed to the new limitations presently in the claims; the new limitations have been addressed in the rejections above.

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL.** See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Allison M. Ford whose telephone number is 571-272-2936. The examiner can normally be reached on 7:30-5 M-Th, alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Michael Wityshyn can be reached on 571-272-0926. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Allison M Ford
Examiner
Art Unit 1651